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=> S (human or sapien) (3A) (kallikrein)
L1        5997 (HUMAN OR SAPIEN) (3A) (KALLIKREIN)

=> S L1 (8A) (INHIBITOR OR INHIBIT OR INHIBITING OR INHIBITED OR INHIBITION OR
MODULATOR OR MODULATE OR MODULATING OR MODULATED OR MODULATION)
L2         564 L1 (8A) (INHIBITOR OR INHIBIT OR INHIBITING OR INHIBITED OR INHI
               BITION OR MODULATOR OR MODULATE OR MODULATING OR MODULATED OR
               MODULATION)

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L3         445 L1 (6A) (METHOD OR ANALYSIS OR ANALYZE OR ANALYZED OR ANALYZING
               OR TEST OR TESTING OR TESTED OR DETERMINING OR DETERMINATION OR
               DETERMINE OR DETERMINED OR EVALUATE OR EVALUATION OR EVALUATED
               OR EVALUATION)
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L4          32 L2 AND L3

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L5   ANSWER 1 OF 22  CAPLUS  COPYRIGHT 2008 ACS on STN
AN   2005:823838  CAPLUS
DN   143:222436
TI   Use of human kallikrein 2 (KLK2) polypeptides and polynucleotides in drug
     screening and in diagnosis of various disorders
IN   Golz, Stefan; Brueggemeier, Ulf; Geerts, Andreas; Summer, Holger
PA   Bayer Healthcare A.-G., Germany
SO   PCT Int. Appl., 96 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     --------------      ----   --------    -------------------    --------
PI   WO 2005075662        A2    20050818    WO 2005-EP342          20050115
     WO 2005075662        A3    20051103
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT,
             RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
             MR, NE, SN, TD, TG
     EP 1711828           A2    20061018    EP 2005-700936         20050115
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS
     US 20070218047       A1    20070920    US 2007-587452         20070119
PRAI EP 2004-1737         A     20040128
     WO 2005-EP342        W     20050115
AB   The invention provides a method of screening for compds. useful in the
     treatment of hematol. disorders, cardiovascular diseases, cancer,
     inflammation, neurol. diseases, reproduction disorders and urol. diseases,
     which involves the use of human kallikrein 2 (KLK2) polypeptides and
     polynucleotides.  Specifically, the drug screening assays look for the
     ability of a compound to bind said KLK2 polypeptides and polynucleotides,
     and modulate the activity of KLK2.  The invention also provides a
     pharmaceutical composition comprising said KLK2 modulator and its use in
     treatment of disclosed diseases and/or disorders.  The invention relates
     said KLK2 modulator may a small mol., a RNA mol., an antisense
     oligonucleotide, a polypeptide, an antibody or a ribozyme.  The invention
     further provides a method for preparing said pharmaceutical composition
Finally,
     the invention provides a method for diagnosing said diseases and/or
     disorder which involves determining the presence and/or expression of said KLK2
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polynucleotides. In the examples, the invention presented the relative expression of KLK2 mRNA in various human tissues and cells, with the greatest expression being detected in the prostate.

L5 ANSWER 2 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2005:528971 CAPLUS
DN 143:362636
TI An Effective Method for Isolating Human Urinary Kallikrein and Trypsin Inhibitor
AU Wang, Ju; Hong, An; Pu, Hanlin; Chen, Xiaojia
CS Bio-engineering Institute, Jinan University, Guangzhou, Guangdong Province, 510632, Peop. Rep. China
SO Zhongguo Yiyao Gongye Zazhi (2004), 35(7), 401-403
CODEN: ZYGZEA; ISSN: 1001-8255
PB Zhongguo Yiyao Gongye Zazhi Bianjibu
DT Journal
LA Chinese
AB The human urinary kallikrein (hKN) and human urinary trypsin inhibitor (hUTI) are two acidic proteins in the urine. The method of ethanol precipitation together with ZnCl2 colloid precipitation was found to isolate them with higher yield and activities.

L5 ANSWER 3 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2003:239423 BIOSIS
DN PREV200300239423
TI DX-88 a recombinant inhibitor of human plasma kallikrein. Evaluation of efficacy and safety in patients with hereditary angioedema.
AU Williams, T.; Cicardi, M. [Reprint Author]; Bork, K.; Gonzalez-Quevedo, T.; Caballero, T.
CS Dipartimento di Medicina Interna, Universita di Milano, IRCCS Ospedale Maggiore, Milano, Italy
SO Journal of Allergy and Clinical Immunology, (April 2003) Vol. 111, No. 4, pp. 908. print.
Meeting Info.: 60th Anniversary Meeting of the American Academy of Allergy, Asthma and Immunology (AAAAI). Denver, CO, USA. March 07-12, 2003. American Academy of Allergy, Asthma and Immunology.
CODEN: JACIBY. ISSN: 0091-6749.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 21 May 2003
Last Updated on STN: 21 May 2003

L5 ANSWER 4 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:830198 CAPLUS
DN 137:291281
TI Complex of human kallikrein 2 (hK2) and protease inhibitor-6 (PI-6) in prostate tumor tissue and methods of using the complex and its constituents
IN Mikolajczyk, Stephen D.; Saedi, Mohammad S.
PA Hyrbritech Incorporated, USA
SO U.S., 19 pp., Cont.-in-part of U.S. 6,284,873.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6472143	B1	20021029	US 2000-667288	20000922

	US 6284873	B1	20010904	US 1999-266957	19990312
	US 20010031860	A1	20011018	US 2001-882648	20010615
PRAI	US 1999-266957	A2	19990312		

AB The present invention provides a novel complex of hK2 and PI-6 and methods of using the novel complex and its constituents. The novel complexes of hK2 and PI-6 of the present invention and the PI-6 exist at an elevated level in prostate cancer tissues. PI-6 also exists at an elevated level in other types of cancer cells. Therefore, the hK2-PI6 complexes and PI-6 of the present invention may be used as a serum marker for detecting cancers, particularly prostate cancer. They may also be used as an immunohistol. marker to detect prostate cancer tissues. In accordance with the present invention, the hK2-PI6 complexes of the present invention may be detected in patient tissue samples by immunohistochem. and/or in patient fluid samples by in vitro immunoassay procedures. Diagnostic methods for detecting the existence of prostate cancer is also provided.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2000:666861 CAPLUS
DN 133:250893
TI A novel complex of human kallikrein 2 (hK2) and protease inhibitor-6 (PI-6) in prostate tumor tissue and methods of using the complex for detecting prostate cancer
IN Mikolajczyk, Stephen; Saedi, Mohammad
PA Hybritech Incorporated, USA
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000055308	A1	20000921	WO 2000-US1937	20000126
	W: AU, CA, JP				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 6284873	B1	20010904	US 1999-266957	19990312
	CA 2365540	A1	20000921	CA 2000-2365540	20000126
	EP 1159411	A1	20011205	EP 2000-905737	20000126
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002538818	T	20021119	JP 2000-605726	20000126
	AU 778038	B2	20041111	AU 2000-27376	20000126
	US 20010031860	A1	20011018	US 2001-882648	20010615
PRAI	US 1999-266957	A	19990312		
	WO 2000-US1937	W	20000126		

AB The present invention provides a novel complex of hK2 and PI-6 and methods of using the novel complex. The novel complexes of hK2 and PI-6 of the present invention exist at an elevated level in prostate cancer tissues. Therefore, the hK2-PI6 complexes of the present invention may be used as a serum marker for detecting prostate cancer. They may also be used as an immunohistol. marker to detect prostate cancer tissues. In accordance with the present invention, the hK2-PI6 complexes of the present invention may be detected in patient tissue samples by immunohistochem. and/or in patient fluid samples by in vitro immunoassay procedures. Diagnostic kits and diagnostic methods for detecting the existence of prostate cancer are also provided.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 22 MEDLINE on STN DUPLICATE 1
AN 1999384018 MEDLINE
DN PubMed ID: 10454435
TI Kallikrein gene delivery inhibits vascular smooth muscle cell growth and neointima formation in the rat artery after balloon angioplasty.
AU Murakami H; Yayama K; Miao R Q; Wang C; Chao L; Chao J
CS Department of Biochemistry and Molecular Biology, Medical University of South Carolina, Charleston, SC, USA.
NC HL-29397 (United States NHLBI)
HL-52196 (United States NHLBI)
SO Hypertension, (1999 Aug) Vol. 34, No. 2, pp. 164-70.
Journal code: 7906255. ISSN: 0194-911X.
CY United States
DT (COMPARATIVE STUDY)
(IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 199909
ED Entered STN: 25 Sep 1999
Last Updated on STN: 3 Mar 2000
Entered Medline: 15 Sep 1999
AB Tissue kallikrein cleaves kininogen substrate to produce vasoactive kinin peptides that have been implicated in the proliferation of vascular smooth muscle cells (VSMCs). To explore potential roles of the kallikrein-kinin system in vascular biology, we evaluated the effects of adenovirus-mediated human kallikrein gene delivery on the growth of primary cultured VSMCs and in balloon-injured rat artery in vivo. Kallikrein gene transfer into cultured rat VSMCs resulted in time-dependent secretion of recombinant human tissue kallikrein and inhibition of cell proliferation. Balloon angioplasty reduced endogenous rat tissue kallikrein mRNA and protein levels at the injured site. In rats that received adenovirus-mediated human kallikrein gene delivery, we observed a 39% reduction in intima/media ratio at the injured vessel after delivery compared with that of rats that received control virus (n=8, P<0.01). Icatibant, a specific bradykinin B(2) receptor antagonist, blocked the protective effect and reversed the intima/media ratio to that of the control rats (n=5, P<0.01). After gene delivery, human kallikrein mRNA was identified at the injured vessel and a 3-fold increase occurred in kininogenase activity. cAMP and cGMP levels in balloon-injured aorta increased significantly at 4, 7, and 14 days after kallikrein gene delivery, but icatibant abolished the increase. These results provide new insights into the role of the vascular kallikrein-kinin system and have significant implications for gene therapy to treat restenosis or atherosclerosis.

L5 ANSWER 7 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1999:717391 CAPLUS
DN 132:104485
TI Purification of human urinary kallikrein from residue of urinary trypsin inhibitor production
AU He, Guozhen; Tang, Liyun; Zheng, Lijun
CS Gungdong Yantang Biochemistry Pharmaceutical Co., Ltd., Canton, 510507, Peop. Rep. China
SO Yaowu Shengwu Jishu (1999), 6(2), 103-106
CODEN: YSJIFO; ISSN: 1005-8915
PB Yaowu Shengwu Jishu Bianjibu
DT Journal
LA Chinese

AB The purification method of human urinary kallikrein (HUKN) combined with urinary trypsin inhibitor was reported. HUKN had been purified by use of CM-Sepharose FF, DEAE-Sepharose FF and Aprotinin-Sepharose CL-4B chromatog. The purified HUKN with a specific activity of 293.9 EU/mg protein and the recovery rate of 48.6% was a single peak on HPLC. The technol. is simple, and can be scaled up easily.

L5 ANSWER 8 OF 22 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN DUPLICATE 2
AN 1995039278 EMBASE
TI Proteinases from the fibrinolytic and coagulation systems: Analyses of binding to pregnancy zone protein, a pregnancy-associated plasma proteinase inhibitor.
AU Earbelaez, L.; Jensen, P.E.H.; Stigbrand, T. (correspondence)
CS Dept. Medical Biochemistry/Biophys., University of Umea, S-901 87 Umea, Sweden.
SO Fibrinolysis, (1995) Vol. 9, No. 1, pp. 41-47.
ISSN: 0268-9499 CODEN: FBRIE7
CY United Kingdom
DT Journal; Article
FS 025 Hematology
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
LA English
SL English
ED Entered STN: 8 Mar 1995
Last Updated on STN: 8 Mar 1995
AB The inhibitory effects of pregnancy zone protein (PZP) on proteinases within the fibrinolytic and coagulation systems have been studied and compared to that of human α(2)-macroglobulin (α(2)-M). Plasmin, t-PA, urokinase, thrombin, human plasma kallikrein, human and porcine tissue kallikrein were tested for binding to PZP and α(2)-M. PZP was cleaved at the 'bait' region as seen in SDS-PAGE, by both human and porcine tissue kallikrein, but not by any of the other proteinases tested and we therefore suggest that PZP may have a role in inhibition of tissue kallikrein. Plasmin, thrombin, and plasma kallikrein were found to be bound and inhibited by α(2)-M by cleavage of the 'bait' regions. Minor amounts of cleavage products of α(2)-M were detected with t-PA and urokinase after prolonged incubation at room temperature. Cleavage of α(2)-M was detected following incubation with porcine tissue kallikrein, but no cleavage was seen following incubation with human tissue kallikrein. The fast inhibition of plasmin, thrombin, and plasma kallikrein suggest that α(2)-M may be physiological relevant as an inhibitor for these proteinases. Despite the similarities between α(2)-M and PZP, significant differences are observed in the inhibition of proteinases. These results suggest distinctive differences in the function of the two human α-macroglobulins, PZP does only seem to inhibit human tissue kallikrein of the proteinases tested from the fibrinolytic and coagulation systems.

L5 ANSWER 9 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1995:196954 CAPLUS
DN 122:127069
OREF 122:23567a,23570a
TI Measurement of urinary kallikrein activity by kininogenase assay
AU Kasatsuki, Tamako
CS Sch. Med., Iwate Med. Univ., Morioka, 020, Japan
SO Iwate Igaku Zasshi (1994), 46(4), 543-52
CODEN: IIZAAX; ISSN: 0021-3284

DT Journal
LA Japanese
AB A method for measurement of urinary kallikrein activity in patients was developed with essential hypertension by kininogenase assay, using com. available reagents. Optimum pH of the activity was 8.5, and Michaelis-Menten constant (Km) value for the activity in bovine low-mol.-weight kininogen was 60 μg kinin/mL and these values were similar to those obtained from purified kallikrein, suggesting that the kininogenase activity represents kallikrein activity. The coefficient of variation was 4.7% for intra-assay and 8.4% for inter-assay, thereby indicating a good reproducibility. Addition of NaCl or albumin into urine sample did not disturb the activity, suggesting that this assay system is available for measurements of the activity in urine obtained from patients with nephrotic syndrome or high Na intakes. When urinary kallikrein excretion was measured by this method in 4 patients with primary aldosteronism, it significantly decreased after excision of adrenal adenomas. This method is therefore useful to assess the precise levels of kallikrein excretion.

L5 ANSWER 10 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1993:163754 CAPLUS
DN 118:163754
OREF 118:27949a,27952a
TI Immunoassays for the determination of human tissue kallikrein (TK) in different body fluids based on monoclonal antibodies
AU Witzgall, K.; Godec, G.; Shimamoto, K.; Fink, E.
CS Dep. Clin. Chem., Univ. Munich, Munich, D-8000/2, Germany
SO Agents and Actions Supplements (1992), 38(1, Recent Prog. Kinins: Biochem. Mol. Biol. Kallikrein-Kinin Syst.), 153-8
CODEN: AASUDJ; ISSN: 0379-0363
DT Journal
LA English
AB A monoclonal antibody produced against human tissue kallikrein was used to develop solid-phase immunoassays for the determination of total immunoreactive tissue kallikrein, the complex of tissue kallikrein with α1-proteinase inhibitor (α1-antitrypsin), and enzymically active tissue kallikrein. The assays permit the specific determination of various forms of tissue kallikrein in body fluids and should be very useful in studies on the biol. function of tissue kallikrein-kinin systems.

L5 ANSWER 11 OF 22 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
AN 1990067249 EMBASE
TI Determination of active human urinary kallikrein and human urokinase (M(r) 33,000 and M(r) 54,000 species) concentration in the presence of cognate serine proteinase.
AU Ascenzi, P.; Amiconi, G.; Menegatti, E.; Guarneri, M.
CS C.N.R., Center for Molecular Biology, Department of Biochemical Sciences, University of Rome 'La Sapienza', Piazzale Aldo Moro 5, 00185 Rome, Italy.
SO Clinical Chemistry and Enzymology Communications, (1990) Vol. 2, No. 2, pp. 79-85.
ISSN: 0892-2187 CODEN: CCECEY
CY United Kingdom
DT Journal; Article
FS 029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991

AB A spectrophotometric method for the determination of active human urinary kallikrein concentration, in the presence of human urokinase (M(r) 33,000 and M(r) 54,000 species), as well as of active human urokinase (M(r) 33,000 and M(r) 54,000 species) concentration, in the presence of human urinary kallikrein, is reported. This rate assay method has been developed from the quantitative analysis of kinetics for the hydrolysis of the N-α-carbobenzoxy-L-lysine p-nitrophenyl ester catalyzed by human urinary kallikrein, in the presence of human urokinase (M(r) 33,000 and M(r) 54,000 species) selectively inhibited by the p-carbethoxyphenyl ester of the ε-guanidino-caproic acid methanesulphonate, and by human urokinase (M(r) 33,000 and M(r) 54,000 species), in the presence of human urinary kallikrein selectively inhibited by the bovine basic pancreatic trypsin inhibitor (Kunitz-type inhibitor). The active enzyme concentration has been estimated, under conditions where [S](0) > >K(m), from the dependence of the initial rate for substrate hydrolysis (i.e., V) on the enzyme concentration (i.e., [E](0)) taking into account the proportionally constant k(cat) (i.e., V = k(cat).ovrhdot.[E](0)). The minimum active human urinary kallikrein and human urokinase (M(r) 33,000 and M(r) 54,000 species) concentration which may be evaluated from the analysis of the experimental data corresponds to 3.8 x 10(-3) μM and 1.4 x 10(-2) μM, respectively.

L5 ANSWER 12 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1989:168941 CAPLUS
DN 110:168941
OREF 110:27917a,27920a
TI Use of 125I-aprotinin in the assay of glandular kallikreins
AU Ryan, James W.
CS Dep. Med., Univ. Miami, Miami, FL, 33101, USA
SO Methods in Enzymology (1988), 163(Immunochem. Tech., Pt. M), 160-9
CODEN: MENZAU; ISSN: 0076-6879
DT Journal
LA English
AB The use of 125I-labeled aprotinin as a tracer for labeled inhibitor-enzyme immunoassay of kallikrein of human urine (and other tissues) is described. The assay measures only that fraction of enzyme capable of binding the inhibitor with high affinity; enzyme-bound inhibitor is separated from free inhibitor by the addition of excess immobilized antibodies monospecific for the enzyme.

L5 ANSWER 13 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1989:130909 CAPLUS
DN 110:130909
OREF 110:21515a,21518a
TI Correlation of two different assays for urinary kallikrein in normotensive and hypertensive subjects
AU Maier, Manfred; Jerabek, Ingrid; Reissert, Guenther; Hoeltzl, Eva; Binder, Bernd R.
CS Dep. Med. Physiol., Univ. Vienna, Vienna, Austria
SO Clinica Chimica Acta (1988), 178(2), 127-39
CODEN: CCATAR; ISSN: 0009-8981
DT Journal
LA English
AB Possible differences in structure-function relationship of urinary kallikrein between normotensive and hypertensive individuals were analyzed using 2 different assay systems which detect 2 distinct entities of the enzyme. A monospecific goat anti-human urinary kallikrein antibody was characterized by inhibition studies with the purified active enzyme and by trypsin activation of

endogenous urinary prokallikrein. Anal. of the data revealed that the antibody is directed against active kallikrein by recognizing an epitope which is different from the catalytic site of the enzyme but which is being exposed together with the active site during trypsin activation of the proenzyme. A direct RIA for urinary kallikrein was developed and correlated with the kinin-generating activity of the enzyme by assessing endogenous active and trypsin-activated kallikrein in the urine of normotensive and hypertensive subjects. Significant pos. correlations were found between the 2 assays for both active and total kallikrein in normotensive and hypertensive subjects, and the slopes of the resp. regression lines were identical. These data do not provide evidence for a defective enzyme, a defective activation of the proenzyme, or the presence of an inhibitor of urinary kallikrein in essential hypertension.

L5 ANSWER 14 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1985:74788 CAPLUS
DN 102:74788
OREF 102:11667a,11670a
TI Reagents for immunoassay of α1-proteinase inhibitor-kallikrein complex
PA Sugiura Shinyaku Kaihatsu Kenkyusho K. K., Japan; Maruko Pharmaceutical Co., Ltd.
SO Jpn. Kokai Tokkyo Koho, 7 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 59203958	A	19841119	JP 1983-78158	19830506
PRAI JP 1983-78158		19830506		

AB An enzyme is bound to antihuman α1-proteinase inhibitor antibody and antihuman kallikrein antibody is immobilized on an insol. solid carrier to prepare reagents for use in the immunoassay of α1-proteinase inhibitor-kallikrein complex in human body fluids by the sandwich method. Thus, α1-proteinase inhibitor and kallikrein in Freund's complete adjuvant were injected into rabbits to form the resp. antibodies. Antikallikrein antibody (40 mg) in 0.1M Na phosphate buffer containing 50 mM NaCl was incubated with polystyrene beads (.apprx.6.5-mm diameter) at 30° for 2 h and then with the same buffer containing 0.1% bovine serum albumin at 30° for 2 h, and the reaction product was kept in the same buffer containing 0.01% thiomersal. Sep., 1.5 mg anti-α1-proteinase inhibitor antibody in 0.1M Na phosphate buffer containing 50 mM NaCl was incubated with 2% m-maleimidobenzoyl N-hydroxysuccinimide ester in dioxane at 30° for 1 h. After separation on a Sephadex G 25 column, the derivatized antibody was treated with 1.5 mg β-galactosidase at 30° for 1 h to form β-galactosidase-labeled antibody. A 20-μL serum sample with addition of 10 mM phosphate buffer containing bovine serum albumin, NaN3, MgCl, and NaCl was incubated with the polystyrene beads at 37° for 2 h, followed by incubation with β-galactosidase-labeled antibody. The beads in the buffer were treated with 0.3 mM 4-methylumbelliferyl-β-galactoside at 30° for 5-20 min, mixed with 0.1M glycine-NaOH buffer (pH 10.3), and the reaction mixture was analyzed at 450 nm with excitation at 360 nm for measurement of galactosidase for the determination of α1-proteinase inhibitor-kallikrein complex.

L5 ANSWER 15 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1984:231910 BIOSIS
DN PREV198477064894; BA77:64894

TI STUDIES OF ACTIVATED COMPLEMENT C-1 INACTIVATOR PLASMA KALLIKREIN COMPLEX IN PURIFIED SYSTEMS AND IN PLASMA QUANTIFICATION BY AN ENZYME LINKED DIFFERENTIAL ANTIBODY IMMUNO SORBENT ASSAY.
AU LEWIN M F [Reprint author]; KAPLAN A P; HARPEL P C
CS DIV HEMATOLOGY-ONCOLOGY, DEP MED, NY HOSP CORNELL MED CENTER, NEW YORK, NY 10021, USA
SO Journal of Biological Chemistry, (1983) Vol. 258, No. 10, pp. 6415-6421. CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
FS BA
LA ENGLISH
AB An enzyme-linked immunosorbent assay (ELISA) was developed for the quantification of C.hivin.1 inactivator-kallikrein (C.hivin.1In-K) complexes. The formation of complexes assayed by this method parallelled the inhibition of [human] plasma kallikrein esterase activity by C.hivin.1 inactivator in purified systems. C.hivin.1In-K complexes were detected when a final concentration of 5.7 nM plasma kallikrein was added to plasma, equivalent to the activation of 1% of the plasma prekallikrein. Exogenous Hageman factor fragment added to plasma induced the rapid formation of C.hivin.1In-K complexes, whereas there was an appreciable delay when the plasma contact system was activated by the addition of kaolin. In both systems, the rate of formation and final amount of complex generated were directly related to the concentration of Hageman factor fragment or of kaolin added, indicating that this proteolytic pathway is tightly regulated. C.hivin.1In-K complexes were not generated by kaolin in plasma congenitally deficient in Hageman factor or prekallikrein or by kallikrein in hereditary angioedema plasma deficient in C.hivin.1 inactivator, thus confirming the specificity of the assay. Sucrose gradient ultracentrifugation studies showed plasma C.hivin.1In-K complexes to have a MW consistent with a 1:1 molar complex. In contrast, the complex displayed an anomalously high MW on gel filtration chromatography. A sensitive and specific probe was developed for documenting plasma kallikrein activation.

L5 ANSWER 16 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1983:402149 CAPLUS
DN 99:2149
OREF 99:443a,446a
TI Biochemistry of human tissue kallikrein
AU Geiger, R.; Hofmann, W.; Franke, M.; Baur, X.
CS Chir. Klin. Innenstadt, Univ. Munchen, Munich, Fed. Rep. Ger.
SO Advances in Experimental Medicine and Biology (1983), 156A(Kinins-3, Pt. A), 275-88
CODEN: AEMBAP; ISSN: 0065-2598
DT Journal
LA English
AB Kallikrein (I) from human urine, pancreas, large intestine, blood plasma, and seminal plasma were studied. Procedures for the isolation of I from these sources are reviewed; amino acid compns. and mol. wts. were determined The various I forms have similar properties with respect to cleavage of synthetic substrates, kinin-liberating and blood pressure-lowering potency, aprotinin complex formation, and inhibition by diisopropylflurophosphate. A sensitive and specific immunoassay for urinary I was developed which used human anti-I IgG and peroxidase; the procedure could be used for determination of other human tissue I levels.
Urinary
I was inhibited progressively by increasing concns. of purified human α1-antitrypsin and serum. Serum from a patient with Z/Z α1-antitrypsin deficiency did not inhibit I; apparently, α1antitrypsin is the only glandular I inhibitor in serum.

L5 ANSWER 17 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1984:97758 BIOSIS
DN PREV198427014250; BR27:14250
TI INHIBITION OF HUMAN PLASMA KALLIKREIN BY COMPLEMENT C-1 ESTERASE INHIBITOR DETERMINATION OF KINETIC MECHANISM AND RATE CONSTANTS.
AU NILSSON T [Reprint author]
CS DEP CLINICAL CHEMISTRY, UMEA UNIV HOSP, S-901 85 UMEA, SWEDEN
SO Thrombosis and Haemostasis, (1983) Vol. 50, No. 1, pp. 229.
Meeting Info.: 9TH INTERNATIONAL CONGRESS ON THROMBOSIS AND HEMOSTASIS, JULY 4-8, 1983. THROMB HEMOSTASIS.
CODEN: THHADQ. ISSN: 0340-6245.
DT Conference; (Meeting)
FS BR
LA ENGLISH

L5 ANSWER 18 OF 22 MEDLINE on STN DUPLICATE 3
AN 82153809 MEDLINE
DN PubMed ID: 6917564
TI Simple chromogenic peptide substrate assays for determining prekallikrein, kallikrein inhibition and kallikrein "like" activity in human plasma.
AU Gallimore M J; Friberger P
SO Thrombosis research, (1982 Feb 1) Vol. 25, No. 3, pp. 293-8.
Journal code: 0326377. ISSN: 0049-3848.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198205
ED Entered STN: 17 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 21 May 1982

L5 ANSWER 19 OF 22 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
AN 1981233530 EMBASE
TI Inhibition of human tissue (urinary) kallikrein by sera of patients suffering from hereditary α(1)-antitrypsin (α(1)-proteinase inhibitor) deficiency.
AU Geiger, R.; Koenig, G.; Fruhmann, G.
CS Abt. Klin. Chem., Univ. Munchen, Germany.
SO Hoppe-Seyler's Zeitschrift fur Physiologische Chemie, (1981) Vol. 362, No. 7, pp. 1013-1015.
ISSN: 0018-4888 CODEN: HSZPAZ
CY Germany
DT Journal; Article
FS 029 Clinical and Experimental Biochemistry
LA English
SL German
ED Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991
AB Sera of patients who present a hereditary alpha(1) antitrypsin deficiency were tested as to their capacity to inhibit human tissue (urinary) kallikrein. The degree of the progressive inhibition was dependent on the nature of the genetically conditioned alpha(1) antitrypsin deficiency. Sera of homozygote alpha(1) type Z/Z showed no inhibitory action; sera of heterozygote type S/Z a diminished inhibitory action in comparison with normal sera.

L5 ANSWER 20 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1982:170697 BIOSIS
DN PREV198273030681; BA73:30681
TI CLINICAL IMPORTANCE OF EXAMINING BLOOD KININS AND INSULIN IN PANCREATITIS.
AU ORLOV V A [Reprint author]; VEDROVA N N
CS DIV HOSP THER SUBORD, BASE CLIN HOSP NO 20, MOSCOW, USSR
SO Terapevticheskii Arkhiv, (1981) Vol. 53, No. 2, pp. 45-48.
CODEN: TEARAI. ISSN: 0040-3660.
DT Article
FS BA
LA RUSSIAN
AB The content of prekallikrein, the kallifkrein inhibitor and kallikrein, as well as the spontaneous esterase activity, the content of kininogen, the blood kininase activity and the pancreatic incretory function were studied in 104 patients with various forms of pancreatitis. The studies were based on the results of the i.v. glucose tolerance test and examination of the immunoreactive insulin content in the blood. During the acute stage of pancreatitis, the kininogenesis was intensified because of disturbed equilibrium in the kalikrein/kallikrein inhibitor system, while the level of kininogen (the kinin precursor) was lowered. Disturbances of the pancreatic incretory function were revealed almost in 90% of the patients with pancreatitis. The program of examining patients with pancreatitis should thus include examinations of the blood kinin system and insulin.

L5 ANSWER 21 OF 22 MEDLINE on STN DUPLICATE 4
AN 77140856 MEDLINE
DN PubMed ID: 14935
TI Kallikrein inhibitors in rat plasma.
AU Hojima Y; Isobe M; Moriya H
SO Journal of biochemistry, (1977 Jan) Vol. 81, No. 1, pp. 37-46.
Journal code: 0376600. ISSN: 0021-924X.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197705
ED Entered STN: 13 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 25 May 1977
AB The kallikrein inhibitor contents of human and animal plasma were determined with glandular kallikreins [EC 3.4.21.8]. One ml of plasma could inactivate 20-700 kallikrein units (KU). Rat plasma was the most potent and inactivated 230-700 KU. However, no enzyme capable of inactivating kallikrein could be found in this plasma. Two fractions which inhibited hog pancreatic kallikrein, a fraction corresponding to alpha2-macroglobulin and a fraction which was eluted prior to albumin, were separated from rat plasma by Sephadex G-200 gel filtration. The former inhibitor could inhibit hog pancreatic kallikrein action on Nalpha-benzoyl-L-arginine ethyl ester (BAEE) as well as in the dog vasodilator assay. The other inhibitor was partially purified from rat plasma. One mg of the preparation inhibited 67 KU and the hydrolysis of 5.8 micronmoles/min of BAEE by hog pancreatic kallikrein [EC 3.4.21.8]. The inhibitor also inhibited other glandular and plasma kallikreins, trypsin [EC 3.4.21.4], alpha-chymotrypsin [EC 3.4.21.1], etc. The optimal pH of the inhibitor was 7.5-8. The inhibitor was unstable below pH 5, and was destroyed by heating at temperature above 60 degrees. The isoelectric point of the inhibitor was determined by Ampholine focusing to be 4.4, and its molecular weight was estimated to be 73,000 by Sephadex G-100 and G-150 filtrations. Several experimental results

suggested that this inhibitor differed from alpha1-antitrypsin.

L5 ANSWER 22 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1978:4252 CAPLUS
DN 88:4252
OREF 88:763a,766a
TI Determination of "contact" prekallikrein and kallikrein inhibitor in human blood plasma
AU Veremeenko, K. N.; Volokhonskaya, L. I.; Kizim, A. I.; Lositskaya, V. M.; Meged, N. F.; Gontar, A. M.
CS Kiev. Nauchno-Issled. Inst. Stolaringol., Kiev, USSR
SO Kininy Kininovaya Sist. Krovi (1976), 62-3. Editor(s): Paskhina, T. S.; Men'shikov, V. V. Publisher: Pervyi Mosk. Med. Inst., Moscow, USSR. CODEN: 36SHAQ
DT Conference
LA Russian
AB Plasma prekallikrein was activated by contact with SiO2 or kaolin and the resulting proteolytic activity (determined by the release of arginine from protamine sulfate) could be inhibited by Trasylol or its analogs, soybean trypsin inhibitor, and α2-macroglobulin but was not affected by ovomucoid. Thus, the activity is due to kallikrein and not to plasmin. In the blood of patients with acute hepatitis the levels of kallikrein inhibitors and prekallikrein decreased below normal. The proteins, apparently formed in liver, can be used as sensitive indicators of hepatic function. In acute pancreatitis the levels of prekallikrein decreased and those of kallikrein and antitrypsin as well as antitrypsin-to-α2-macroglobulin ratio increased above normal. After treatment the ratio decreased. The contact determination of prekallikrein and kallikrein inhibitors is simple and reproducible and therefore suitable for clin. use.

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